infarction on arrival, and that patient did receive thrombolytic therapy when the ECG diagnosis was reviewed. Therefore, 4.2% of all patients triaged out of CCU had MI. The accuracy in diagnosing MI in the triage room therefore, was 95.8%.

Conclusion: The introduction of CCU triage was associated with a small number of infarct patients bypassing CCU. In practice, only one of the 43 patients (2.3%) would have had earlier management by CCU admission. This would have meant 592 additional patients being admitted to CCU.

933-60 A Randomized Single Blind Trial of 2-hour Regimens of Alteplase and Streptokinase in Acute Massive Pulmonary Embolism

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In massive pulmonary embolism (MPE), a 2-h regimen of streptokinase (STK) may induce as fast hemodynamic improvement as a 2-h regimen of alteplase (t-PA). 60 Pts with MPE, defined as Miller score > 17/34 and mean pulmonary artery pressure (MPA) > 20 mmHg were randomly allocated on a 1/2 basis, either to a 100 mg/2 h infusion of t-PA (21 Pts) or to a 1.5 million IU/2 h of STK (36 Pts). MPA, cardiac output (CO), and total pulmonary resistance (TPR) were serially assessed over a 12-h period using a 5-way Swan-Ganz catheter. Pulmonary angiography was performed before thrombolytic therapy (TT) and perfusion lung scan 36 h later. Relative changes in TPR over 12 h following onset of TT were:

	T1/2h	T1h*	T2h	T6h	T12h
t-PA	−20 ± 13%	−32 ± 12%	−37 ± 18%	−42 ± 19%	−45 ± 15%
STK	−14 ± 18%	−21 ± 19%	−28 ± 20%	−40 ± 22%	−48 ± 20%

A significant decrease in TPR occured in both TT groups, but more rapidly in the t-PA group compared to the STK group at 1 h (*: p = 0.01). However, the difference was no longer significant at 2 h, after completion of both TT infusions. The mean change in perfusion lung scans at 36 h was similar in both groups.

Conclusion: Improvement of TPR was achieved faster with a 2-h regimen of t-PA compared with a 2-h regimen of STK. However, catch-up phenomenon occured at the end of thrombolytic infusion.

933-61 Prehospital Thrombolysis After Cardiopulmonary Resuscitation in Suspected Myocardial Infarction

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Cardiopulmonary resuscitation (CPR) is usually considered to be a contraindication for iv-thrombolysis (TL) in acute myocardial infarction (AMI). Since data on outcome of prehospital initiation of TL after CPR are limited, we reviewed all consecutive missions of our physician-escorted mobile coronary care unit from Jan 1992 until Dec 1995. *Results:* A total of 59 pts (age 62 ± 14 years, 64% male) with suspected AMI underwent prehospital TL (streptokinase n = 58, urokinase n = 1) after CPR. Fourteen pts had inferior, 37 anterior, 8 AMI of unclear localisation (bundle branch block). Median time from symptom-onset to CPR was 35 min and to initiation of TL 60 min. At alarm of resuce service 19 of the pts had already collapsed. In 8 pts CPR was initiated by a bystander, in 19 by an EMT, and in 32 by the emergency physician. Seventeen pts died on scene without apparent bleedings. Of the 42 pts admitted to a hospital, the cause of cardiac arrest was an AMI in 34 and pulmonary embolism in 2. In 5 pts an AMI could not be documented due to missing data in pts dying soon after admission. Of the 42 admitted pts, 29 (69%) were discharged. Thirteen pts died in the hospital (4 of these regained temporary consciousness): 9 died from a cardiac cause, 2 from bleedings, 2 had persisting coma. Beside the 2 lethal bleedings (1 intracerebral, 1 of unclear localisation with shock), severe bleedings were observed in 4 other pts. Three pts required transfusions (2 gastrointestinal, 1 oropharyngeal bleedings), 1 had a pharynx hematoma. Seven pts had minor bleedings not requiring therapy. In a multivariate analysis a CPR duration of ≥20 min was the only factor midly related to the risk of a severe bleeding complication (p = 0.07) *Conclusions:* Prehospital TL after CPR in pts with suspected AMI is associated with a promising short-term outcome but a moderate bleeding risk. Further studies are needed to determine long-term prognosis associated with this therapeutic option.

933-62 Transthoracic Defibrillation: Does Electrode Position Alter Transthoracic Impedance?

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Successful termination of ventricular fibrillation by transthoracic shocks is dependent on achieving adequate current flow, which in turn is governed by transthoracic impedance (TTI). The American Heart Association (AHA) Advanced Cardiac Life Support textbook recommends three electrode positions for defibrillation: 1) anterior/apex, 2) apex/posterior and 3) anterior/posterior. However, there are few data available comparing TTI of these positions. To study this, we applied large (78 cm^2) self-adhesive monitor-defibrillator pads to 20 subjects (10 male, 10 female, ages 21–79) and measured TTI using a validated test-pulse technique which does not require actual shocks. The electrode pads were applied in the three positions recommended by the AHA. All TTI measurements were made at end-expiration and body surface area (BSA) was recorded.

Results: (mean ± SD)

	Position:			p
	anterior/apex	apex/posterior	anterior/posterior	
TTI (ohms):	82.0 ± 24.7	71.2 ± 23.5	77.0 ± 24.7	NS

Correlation of TTI (anterior/apex placement) with BSA: TTI = 15.9 (BSA) + 46.7, r = 0.60, $p < 0.01$; the correlations of TTI and BSA were similar in the other two electrode positions. Thus, the three AHA-recommended electrode positions for transthoracic defibrillation have equivalent and acceptable TTI's; current flow should be similar using any of these positions. TTI is related to BSA in any of the three recommended positions; patients with high BSA and TTI may require higher energy selection to achieve defibrillation.

933-63 Do Transthoracic Shocks Impair LV Function in Humans?

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It has been assumed that high energy DC shocks transiently depress LV performance. However, the independent effect of the shock (vs. VF and its metabolic sequelae) on ventricular function has not been systematically assessed in humans. Therefore, we studied the effects of a series of 3 synchronized "defibrillation strength" transthoracic shocks (200J, 200J and 360J) given at 60 second intervals during sinus rhythm on measures of LV chamber size and function derived from transesophageal echocardiography in 5 pts. Short axis echocardiographic images were obtained at the mid-papillary level and an area-length method was used to calculate LV volumes. In addition, the short axis image was divided into sextants, and regional wall thickening (RWT) was measured. *Results:* All pts had coronary artery disease and diminished LV function (LVEF: 14%–30%). The sequential shocks did not affect heart rate or systemic blood pressure and did not consistently alter stroke volume (SV), cardiac output (CO), LVEF, or RWT (in all 30 sextants) [all p > 0.1 by ANOVA]. Detailed analysis of RWT revealed significantly increased thickening in the worst baseline sextant (p = 0.05), but a tendency for RWT to worsen in the best sextant.

Mean ± SD	Control	200J-1	200J-2	360J
SV (ml)	65 ± 22	56 ± 17	60 ± 11	55 ± 24
CO (L/min)	4.3 ± 1.0	3.7 ± 0.9	3.9 ± 1.0	3.5 ± 1.4
LVEF (%)	22 ± 7	19 ± 6	20 ± 6	20 ± 11
RWT (mm)	0.9 ± 1.2	1.1 ± 1.3	1.3 ± 1.3	1.2 ± 1.6

However, in one pt, LVEF and CO fell from 16% to 7% and 3.4 to 1.3 L/min by the final shock.

Conclusions: Defibrillation strength transthoracic shocks do not consistently impair LV performance in pts with coronary artery disease and compromised LV function, but the effect is widely variable and, in some pts, clinically significant depression of LV function may occur.